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TASK I - PHASE I

PILOT STUDY OF CANCER MORTALITY NEAR AN ARSENICAL PESTICIDE PLANT IN BALTIMORE

MAY 1976 FINAL REPORT



U.S. ENVIRONMENTAL PROTECTION AGENCY

Office of Toxic Substances 4th and M Streets, S.W. Washington, D.C. 20460

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EPIDEMIOLOGY STUDIES

TASK I - PHASE I

PILOT STUDY OF CANCER MORTALITY
NEAR AN ARSENICAL PESTICIDE PLANT IN BALTIMORE

BY

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EPA Contract No. 68-01-2490 EPA Project Officer: Robert J. Carton, Ph. D.

For

U. S. Environmental Protection Agency Office of Toxic Substances 4th and M Streets, S. W. Washington, D. C. 20460

May 1975

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Acknowledgements

The authors wish to thank Drs. Beetjer, Levin, and Lilienfeld for their suggestions and support as well as for the provision of relevant information which permitted the completion of certain portions of this study. They would also like to thank Dr. C. W. Kruse for providing information on local wind directions.

The measurement of long-term effects from low-level exposure to carcinogens in air has been a difficult problem because of the presence of multiple agents, the changing population bases exposed, and the involvement of personal risk factors during the long latent period before cancer is manifest. Possibly because of these confounding variables, the studies relating levels of air pollution to localized cancer mortality by census tracts have been few in number and frequently unrewarding. Winkelstein and coverkers (1) in Buffalo and Zeidberg, Horton and Landau (2) in Nashville found no correlation between the level of pollution and the risk of lung cancer. However, Menck, Causgrande and Handerson (3) did find higher rates in contaminated areas of Los Angeles which they associated with the presence of benzo(a)pyrene in air.

Studies sixed specifically at determining the bealth effects of arsenic in the general environment, especially around smelters, are also limited. Milham and Strong (4) have demonstrated excessive levels of arsenic in urine and hair of c'ildren exposed to the emissions from a copper smelter but no detrimental health effects were noted. Blot and Fraumeni (5) have reported excess lung cancer mortality for both males and females in counties with copper, lead or zinc smelting industries. The latter data agree with the reported risks of lung cancer in populations occupationally exposed to assenic (6, 7). A direct association of cancer and environmental assenic has not been demonstrated.

The current pilot project tests the feasibility of determining the carcinogenic effects of arsenic by examining the mortality of populations living near an insecticide-producing industry. Cancer death rates in populations from carsus tracts near the plant are compared to all the tracts in the city which match on several variables. The advantage of using this matching study design is that data from other projects with similar designs might be combined despite differences in the characteristics of the populations exposed to the arsenic in various cities.

Background

For many years a chemical plant in south Baltimore produced arsenicals as well as other obswicels for agricultural use. Artenic acid was manufactured in the early 1900's but this operation was discontinued over 20 years ago and the plant reconstructed in 1952. The insecticide portion of the plant produced several arsenates but these chemical procedures have also been terminated. Lead arsenate production was discontinued in 1967, calcium arsenate in 1973 and sodium arsenate in 1974. The plant continues to package many of these arsenicals for distribution. The insecticide plant has also produced chlorinated hydrocarbons and organic phosphates.

The area around the plant is heavily industrialized. Adjacent to the chemical plant was a Jas and Electric Company operation. Rubber production also took place nearby. The significance of these possible sources of contamination must be investigated.

Mechods

Index census tracts with possible environmental exposure to arsenic were defined empirically as those tracts for which at least fifty percent of the area was included within a 3/4 mile radius of the plant (Figure 1). This distance was selected because any smaller radii would have included only the single tract in which the plant was located and larger radii would have included tracts across the river in so area with heavy industry and few private dwellings within one mile of the chemical plant. Four census tracts which fell within these boundaries are 2303, 2302, 2404 and 2301. The populations of the first three tracts were similar in demographic and socioeconomic characteristics (See tables 1 and 2, Figure 2). The 1970 census deta indicated that the residents were an older, stable, white population with median incomes from \$8400-9200 per annum. The population of tract 2301 had a lower median income, a higher proportion of families at poverty level. and a higher parcentage of blacks than the other three tracts. Separate matching control tracts were selected for the first three index tracts. Match I, and for 2301, Match II. All census tracts in Baltimore City which matched the index traces on six variables were chosen as controls. The variables and the range of differences which were acceptable for matching were:

Several of the matching tracts were adjacent to the index tracts and have been analyzed separately in the event that the population of those tracts may also have had minimal exposure. One possible control tract was dropped because of an excess of white females in the older age group resulting from the presence of a nursing home in that tract.

For some of the analyses, the control tracts have been subdivided into groups according to geographic location since the populations in various areas of the city differ in their exposure to heavy industry or in mobility. The southern tracts have several areas of industrialization. The central area includes inner city tracts with both problems of industrial exposure and mobility. The northern tracts are found in primarily residential areas. No Match II controls were found in the south.

All death certificates in Baltimore City for the three years 1970-1972 have been examined for cancer listed anywhere among the causes of death. All cancers have been classified according to the 1965 ISCD code. If no cancer code appeared on the cartificate, the cancer was classified by the staff; otherwise the coding as recorded by tity vital statistics personnel was accepted. The occupation and place of employment as recorded on the certificate were listed in order to detect deaths of chemical plant workers. Such information might also identify other dangerous occupations.

The common tract codes noted on the certificates were used for classifying residence. The validity of the codes was checked by selecting a seven and ten percent systematic sample of the cancer deaths in the years 1971 and 1972 respectively. The census tracts incorrectly coded were eight percent in the first sample and fifteen percent in the second sample. To avoid erroneously adding cancer deaths to index tract mortality from misclassification, the residence was listed for all cencer deaths in the index tracts and recoded correctly. This procedure allowed elimination of deaths from the index tracts but no additions from misclassifications in other tracts, thus providing a minimum estimate of cancer mortality in these tracts.

Results

The crude cancer death rates for selected sites and total cancers in each sex are presented in tables 3 and 4 for the combined tracts. The data suggest that the risk of lung cancer in males is 1.4 times higher in the index as compared to the non-adjacent control tracts. The risk in the mixed racial index tract is even higher in comparison to its matched controls. The lung cancer mortality for females is not higher in the index as compared to the adjacent control tracts.

The races in these tables include cancer as listed enywhere on the death certificate but the relationship is not changed if only underlying cause is used. The inclusion of lung cancers classified as secondary or unspecified does not change these observations.

Although the tracts have been matched by age, race and sex so that crude rates should be comparable in the populations, the sex-specific rates of the white population have been adjusted for age in tables 5 and 6 in order to determine whether this variable had any influence on lung cancer mortality. The 1970 Baltimore City population has been used as the standard for a direct adjustment. In these tables, the matching tracts have been subdivided by their location in northern, central and southern geographic areas in order to detect any possible internal differences in controls. None of these adjustments have altered the conclusions based on the crude rates although the magnitude of the excess risk of lung cancer mortality varies depending on which geographic area control is used.

Race-specific rates adjusted for age and sex in tables 7 and 8 indicate that the blacks in the one index tract 2301 may not have a higher rate than inner city populations after adjustment although the white population will have a higher rate. The number of deaths in blacks is small.

The crude death rates for individual index tracts are presented in rables 9 and 10. The data in table 9 indicate that the differences between lung cancer death rates in white males in tract 2303 where the plant is located and the northern and southern controls are significant for both comparisons with probabilities of .0002 and .0003 respectively (8, 9). The

relative risk weighted by age of lung cancer deaths in males in this treet is 3.3 and 4.0 times higher than in the southern and northern controls respectively. Although the relative risk of lung cancer is higher in males in tracts 2301 and 2302, suggesting a possible gradient in death rates for this disease, the differences between index and control rates are eignificant for 2301 only. The death rates for several other cancer sites (oral, stemach, pancress, and prostate) appear high in one or more of the census tracts near the plant but the numbers of deaths are so small that no importance can be attached to the observation.

It was suspected that employees of the plant who lived in the local area might be contributing to the excess mortality in the index tracts. An examination of the industries of employment recorded on the death certificates revealed one man with lung cancer who worked in a chemical plant. Employment records from the insecticide plant were checked to determine whether any of the cancer deaths had occurred in workers. The same case of lung cancer noted above and a prostatic cancer death were matched to plant employees. Removing the case of lung cancer reduces the rate for tract 2303 from 395 to 355 per 100,000 white males. The significant difference in lung cancer deaths in tract 2303 as compared to controls remains.

The occupation as listed on the death certificate indicated no other work exposure which might explain the excess deaths. It is recognized how inaccurate such data are in determining an individual's occupational history.

Out of the 96 cancer deaths in the index tracts 28 had no reported occupation and an additional 19 were listed as housewives. Among the remaining 49 cancers, 5 occurred in laborers, 4 in machinists, 4 in truck drivers, 3 in salesmen with all other occupations being noted less frequently. The industries recorded on the death certificates also did not indicate any remarkable excesses. Fifty cancer deaths had no industry noted and the 19 housewives are included here. The only industry noted three times was "shipbuilding and repair" which may relate to the presence of a large shippard in the south-eastern corner of the city.

The data were suggestive of a gradient in lung cancer mortality in a northerly direction with intermediate rates in index tracts 2302 and 2301 as compared to the tract with the chemical plant and lower rates in the adjacent control tracts. A spot map of the residences of individuals dying of lung and other cancers during the three year period 1970-72 are noted in Figure 3. These data also suggest a clustering of lung cancer deaths. An examination of the direction of wind currents in Baltimore indicate the winds with highest velocity flow from the west and northwest 32 percent of the time in the winter. These winds should dump most of the contamination into relatively unpopulated areas. The south and southwest winds which could be carrying contamination into the northern tracts occur 22 percent of the time in the summer and only 13 percent of the time in the winter and have a lower velocity. Figure 4 demonstrates wind roses for Dundalk Terminal four decades ago; they are not believed to have changed significantly since that time.

Discussion

A metched control design similar to that utilized in the present pilot study might be effective in determining the cancer risks associated with environmental pollution. Frequently the population exposed to any single source of pollution may be too small and the cancers too infrequent to be sure of significant results. By using the matched census tract design to study several exposure sites even in different cities, one may be able to add the data or at least find consistent results which will support an association between a specific contaminant and a specific cancer even though there are basic differences in the characteristics of the populations exposed at each of the various sites.

In the current pilot project there is a significant increase in lung cancer mortality in males in a census tract adjoining an industrialized area containing an arsenical-producing chemical plant. The data also suggest a decreasing northerly gradient in rates for this cancer which could be related to distance from the plant. Although cancer rates for several other sites may appear high, the numbers are too small to attach any weight to the observation.

The lack of any excess lung cancer mortality in females raises questions about whether the differences should be attributed to environmental or occupational exposure. An examination of ylant records indicates that employment in the chemical plant cannot explain the male-female discrepancy or the excess lung cancer mortality. From death certificate data no other industrial exposure predominated in these men but this occupational information is incomplete and unreliable. It is possible, however, that environmental exposure to agents such as ersenic may require the synergistic action of cigarette smoking before lung caucers are manifest. This hypothesis would be compatible with the data on occupational lung cancer in asbestos and uranium mine workers who experienced an excess of lung cancers only in association with digaratte emoking. Exposure to ersenic in females may not produce lung cancer because of infrequent tigarette smoking in the past, although it may produce cancer at a size other than the lung. Moss (10) reported that female woolen textile workers had an excess risk of oral cancers which he attributed to arsenic exposure from sheep-dip; he did not report an excess risk of lung cancers. Thus, health effects from environmental exposure to an agent may be different depending on sex, age, and simultaneous exposure to other personal risk factors.

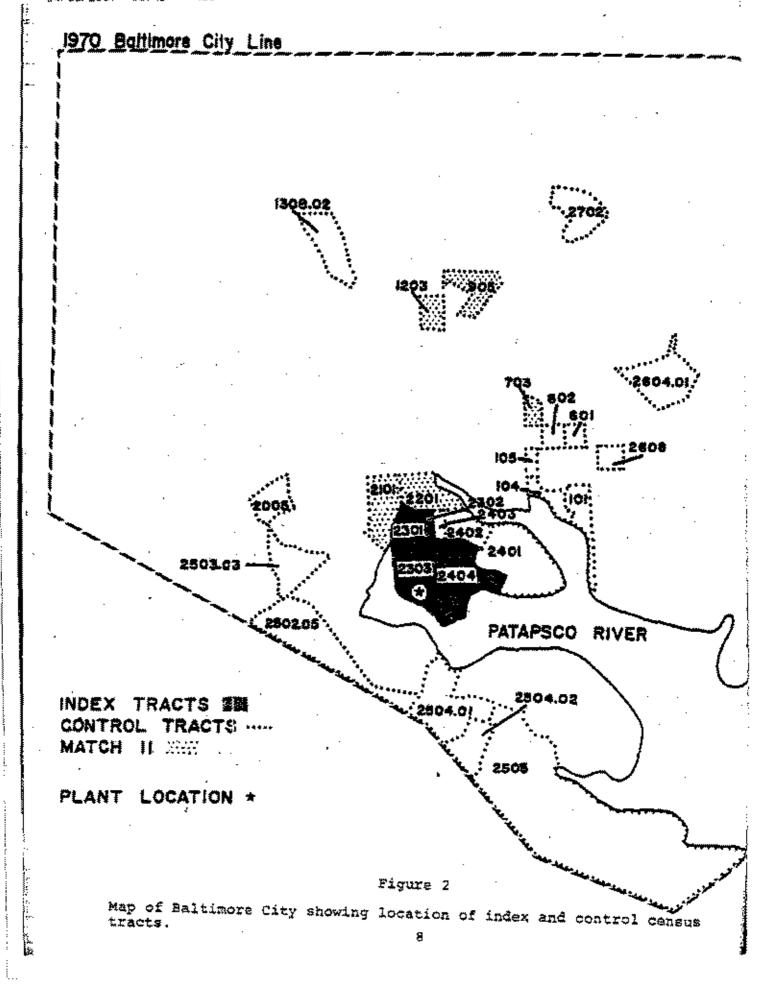
The data from this study are still preliminary. The information should be expanded with deaths from additional years, validation of pathology records and further information on the personal characteristics of the deceased. Sampling of soil and dust in the area to detect the presence of arsenic or other conteminants should be accomplished.

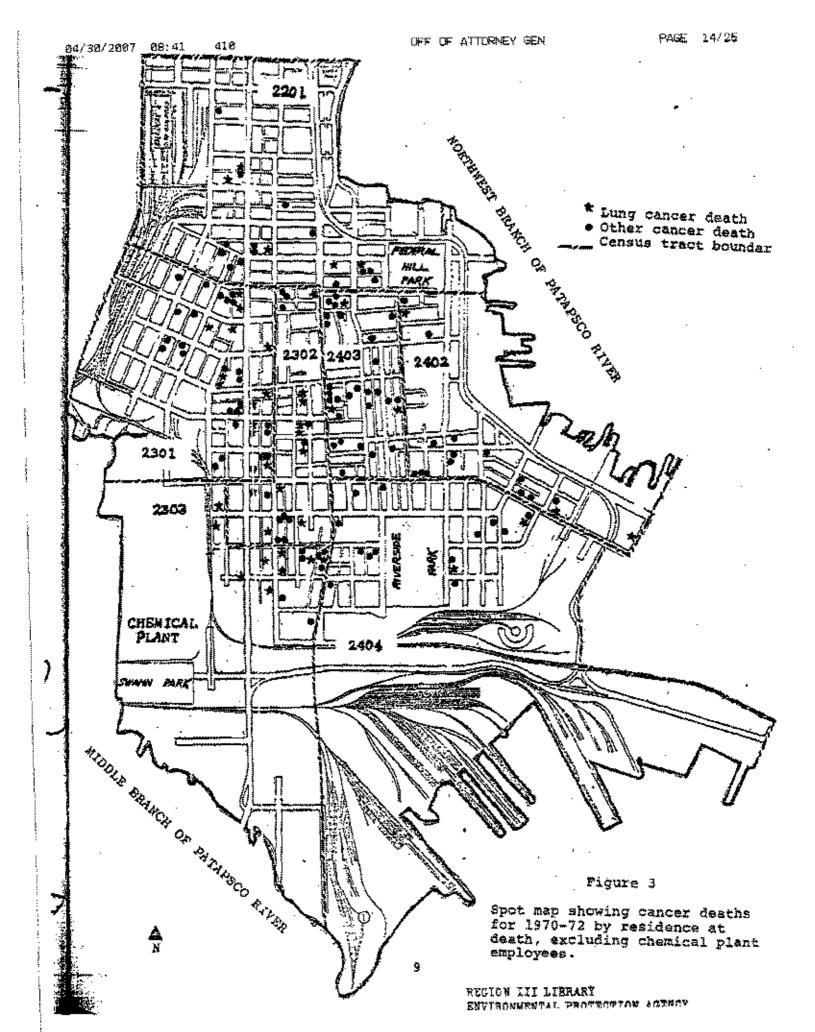
Summary

The present study was a preliminary attempt to determine the Fessibility of using cancer mortality data to indicate the carcinogenic effects of possible arsenic exposure in the environment. The four sensus tracts for which fifty percent of the areas lay within a 3/4 mile radius of an arsenical-producing insecticide plant were matched by such demographic characteristics as age, race, sex, and sociceconomic status with all other similar tracts in Baltimore City. The matching fracts were then subdivided according to suspected differences in exposure to environmental pollution. The lung cancer mortality was higher for the combined tracts adjacent to the plant as compared to the control tracts. The lung cancer mortality for the tract in which the plant was located was three to four times higher than in controls tracts, a difference which was significant. The data also support a northerly gradient in rates with increasing distance from the plant.

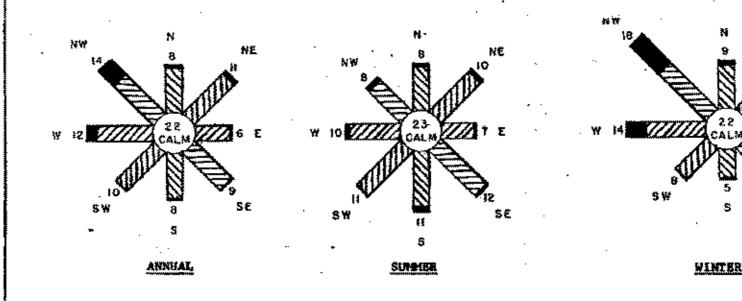
The difference in lung cancer mortality which was present only in males could not be explained by employment in the insecticide plant or in other high risk occupations. It is possible that differences in smoking habits in previous time periods might explain the discrepancy in rates by sex. Additional deaths as well as further information on occupation and personal habits are needed to validate the findings.

PATAPSCO RIVER Wap of area surrounding chemical plant Lydane 1





FEGURE 4



LECEND ZZZZIVELOCITIES 4 THRU 15 MPH VELOCITIES 16 MPH AND OVER VELOCITIES C THRU 3 MPH RECORDED AS CALM. NUMBERS ARE PER CENT OF TIME WINDS BLEW FROM DIRECTION INDICATED

NOTE: WIND ROSES BASED ON ANALYSIS OF MUNICIPAL AIRPORT WHO ROURLY WIND RECORDS (1933 THRU 1938)

WIND DATA BALTIMORE, HD Date: March 1950 Drawn by: THG

Table 1. Designation of census tracts

	index Tracis	Adjacent Controls	South Controls	Central Controls	North Controls
Metch	2302 7303* 2404	2407 2402 2403	2008 2502.08 2503.03 2604.01 2504.02 2505	101 194 195 861 602 2608	300.02 3604.03 2702
Maich ii	230‡	550t 310t		· 703	905 1203

^{*}Plant tocated in this tract.

Table 2. Characteristics of index and control consus freets from 1978 consus for selected Saitimore areas.

TRACTS

!	Matel	h !			Non-adjacent	Non-adjacent	Non-adjacent	Matc		Non-atjacent	Non-adjacent
Characteristics	2340	क्षापट	2414	Adjacent Controls	South Contrais	Contrals	North Controls	2361	<u>Adjament</u> Controls	Controls Controls	Controls
Total Population Range between tracts	1703	3395	3697	9347 2672-8356	31,174 3143-6893	20,978 2126-4655	12,884 2316-5960	3500	5606 1935-3671	318 0	\$139 2962-51 8 7
% < 5 yıs	8	8	ð	6-8	6-12	\$-9	8- fO	†0	8	- 10	9
% 5-19 yrs	29	28	28	25-2 0	24-30	23-98	23-26	30	26-30	30	29-33
% 20-44 yra	28	28	29	26-29	51-00	26-32	26-36	26	25-25	26	38-34
% 45-64 yrs	28	24	25	29-38	14-28	25-33	22-27-	23	25-2 6	23	17-18
% 85+ yrs	\$	11	16	10-16	4-13	12-16	6-17	10	12-13	12	8+10
% mate	50	50	48	48-49	47-49	46-60	47-49	4\$	48-54	· 48	47-48
% black	-	0.4	٠	0.1.0	0.4-2.5	D-1.9	0.1-3.9	50	50-52	47	39-64
Median Income*	8400	9200	8400	#100 to 9300	7500 to 9000	7500 to 9308	8800 to 10000	6509	5800 to 6000	0099	7000 to 7300
% below poverty tevel	8	8	9	7-8	₽-16	7-14	5.7	25	28-29	17	20-22
% head of household 65+ yrs	19	22	19	9-24	9-23	9-36	. 14°10	† †	18-31	20	3-16

[&]quot;Rounded to negreet hundred dollars,

Table 3. Crude death rates for specific types of cancer for total population in index tracts and matched controls for both Match I and Match II, arrange rates per 100,000 per year, total deaths for 1970-72, males only.

			Match I		· .	Metch N.,	
Type of tencer a 8th revision ISC		Index	Adjecont Controis	Non-adjacent Controls	ndex	Adjacent Controls	Non-adjacen Controls
Oral (140-149)	# :#1#	2 15.48	3 21.99	6 6.47	19,11	2 23.6#	0
Stomach (151)	# rate	1 7.73	† 7,33	13 14,02	· c	1 11.84	1 0.20
Calon (153)	# FBFB	2 15.46	3 21.99	\$ ^15 15.18	ç	3 35.52	4 24.86
Rectum (154)	# rate	2 15.45	3 21.99	7 7.65	0 -	. 0	0
Pancraes (157)	# #	2 15.46	¢	10 10.79	1 19.11	† 11.84	0
Lung (162)	# ####	19 146.85 ^b	10 75.31	97 19461 ^b	11 215.24 ⁸	10 118.41	12 74.40 ⁸
Prostate (185)	# rate	\$ 23.19	3 21.99	13 · 14.02	3 57.34	3 35.52	3 18,60
Bladder (168)	# sete	C -	† 7.33	9 9.71	C .	0	; 8.20
Lymphomat (200-203)	# FAţu	7.73	0	11 \$1,86	1 18.11	2 23.68	0
), eukemia (204-207)	# ####	0	ņ	6 8.47	19.11	Ö	3 18.60
All cancer (140-209)	# 78te	38 293.75 b	40 293.23	241 259,82 ^b	24 458.72 ⁴	31 . 357.06	40 248.02 ⁸

a Index significantly different from non-adjacent controls by Woolf-Haldene method (8, 9).

a index not significantly different from non-adjacent controls.

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Table 4. Crude death rates for specific types of cannor for total population in index tracts and matched controls for both Match I and Match II, sverage rates per 100,000 per year, total deaths for 1970-72. (Smales only,

			Match I			Metch II	
Type of cancer 8th revision (5:)		l fluitecc	Adjacent Controls	Non-adjacent Controls	Index	Adjecent Controls	Non-86/scent Controls
Oral (140-148)	# rate	2 14.67	3 20.83	3 2.98	0	1 11.94	Ç.
Stomach (151)	tsle \$	0	3 20.83	8 7,9/3	18.36	1 11.04	`a 16.83
Colon (153)	# rete	7.44	2 13.89	21 20.83	18.38	3. 25.#3	6 33.65
Roctum (1 54)	k e ¢é ₩	0	1 6,94	\$ 7,93	0	ð	
Pancress (157)	# rate	3 22.91	1 8.64	14 13.89	0	2 23.89	2 11.22
Lung (162)	. rate	3 22.31	27.78	15 14.85	1 18.36	3 35.83	2 11,22
Breast (174)	# FMCH	1 7,44	\$ 6.94	27 26.78	5 91.78	3 35,83	6 33.85
Cetvix (180)	# rate	7.44	2 13. 59	5 4. 96	1 18.38	4 47.77	† 5.61
Diedder (188)	# rate	0 -	ō	3 2.88	Đ	1 11.94	2 11.22
Lymphomas (200-203)	# rate	0	1 6.94	10 9.92	0	0	1 4.61
Leukemia (204-207)	. #	· 2 14.87	0	5 4.98	1 18.36	0	0 -
Ail Cancer (140-206)	# rete	18 133.84	25 384,44	177 175.55	16 293.59	23 274.69	33 185.09

Table 5. Death raise for enjected cancer in white male populations of Marich I and white Metch II Index and control census tracts, raise per 100,000 adjusted for age to 1970 Ballimore City white males.

Type of cancer and 6th revision (SCD code	Baltimore .City	Index	Adjacent Controls	South Controls	Central Controls	North Controls
Oral (140-149)	12.05	19.11	22.81	2.53	10.06	9.75
Colon (153)	. 36.34 ⁸	18.71	27.85	24.62	12.24	20.21
Pencress (157)	11.47	73.11	-	14.48	12.71	5.03
Lung (162)	95.62	181.52	\$0,12	144.07	105,13	103,01
Prostate (185)	24,13	37,38	17,43	23.21	16.29	10.11
Lymphomes (2IX)-203)	20.88 ^b	15.89	5.47	18.96	12,43	4.72
Loukemies (204-207)	20.46			8.15	9.67	9.40
All Cancer (140-208)	282,75	608.91	305.76	328.97	301.03	274.56

a This rate incitioes ISCD codes 193 and 154.

Table 8. Death raise for salected campers in while female populations of March 1 and white Match II index and control campus tracts, raise per 100,000 adjusted for age to 1975 Saliknors City white females.

Type of cancer and 8th ravision (SCI) code	Beitimore City	Index	Adjacent Controls	South Controls	Central Contrals	North Controls
Oral (140-149)	4.08	15.80	17.67	5.98	+	5.01
Calon (163)	32.51 ² .	8.84	23.57	24.83	24.27	39.25
Pancress (15?)	9,35	26.63	9.00	21.61	23.89	5.01
Lung (162)	38.43	25.77 •	29.¥8	11.95	18.27	26.81
Bresst (174)	35,87	44.03	18.01	36.40	30.84	47.88
Cervix (180)	7,90	8.88	29.22	7.81	3.28	3.58
Lymphoma# (200-203)	16.08 ^b -		9 ,06	6.89	16.04	14.22
Leukemiz (204-20')	16,05	8,16		15.04	2.72	3.01
All Caricer (140-2(19)	192.96	144.64	240.28	212,08	212.20	277.06

a This rate includes ISOD codes 153 and 154.

a This rate includes ISCD codes 200-209.

b This rate includes ISCD codes 200-209.

Table 7. Death reter for sciented concern in populations of Match I and whites in Match # Index and control cansus tracts, rates per 100,000 adjusted for age and sex to white 1970 Ballimore City population.

Type of cancer and 8th revision ISCD code	Saltimora City	index	Adjacent Controls	South Controls	Central Controls	North Controls
Oral (140-149)	7.85	14.42	20.08	4.35	4.7 5	7,25
Colon (983)	34.32 [#]	13.63	25.5 2	24.62	21.17	30.25
Panoress (157)	18,35	20.28	2.17	18.24	18,61	\$.01
Lung (162)	54.85	99.85	57.98	74.34	58.23	82.79
Lympho:has (200-203)	18.34 ^b	7.50	5.75	11.50	13,86	8.73
Laukemis (204-207)	18,34"	3.25	٠.	9.68	6.00	7.09
All Cencer (140-209)	235.38	363,67	272.81	287.24	284.15	275,89

s This rate includes ISCD codes 153 and 154. b This rate includes ISCD codes 200-209.

Table 8. Death rates for astected cancers in black populations of Martin II leafer and content assesse tracts, rates pre- 100,000 edjusted for age and sex to black 1970 Saltimore City population.

Type of sencer and Bits revision (SGC) code	City City	Index	Adjacent Controls	Central Controls	North Controls
O/al (\$40-149)	4.26	6.07	17.21		
Colon (153)	17.77 ^{8.}	9.97	17.02	68.11	36.61
Pancreas (157)	8.01	9.07	25.63		-
Lung (162)	39.08	72.77	49.28	110.46	35.98
Lymphomaa (200-203)	ь		8.56		#
Leukomia (204-201)	11.03 ^b	31.37	_		17.88
Ali Cancer (140-299)	167.83	236.33	215,82	353.44	208.31

a This rate includes ISCO codes 153 and 154. b This rate includes ISCO codes 200-209.

Table 9. Crade cancer denix rates for selected sites in males by individual tract and matched controls, average somesi death rate per 190,000.

MATCHI

Cancer Site	2303	10dex	2404	Adjacent Controls	South Controls	Central Centrols	North Controls
Orel	39,6	108	+	22.6	2.2	10.3	10.7
Pancreas	38.5	_	18-6	-	11,1	21.9	28.7
Lung	S94.6	138.9 ²	. 37.3 ^b	79.3	109.2	162.6	96.4
Prostate	39.5	19.8	18.0	22.0	tas	17.3	6.4

MATCH

	Instax	2301	Adjacent	Controls	Contral	Controls	North (Controls
Cancer Site	With	₽M	W#A	3M	WW.	BM	WM	BM
Orej	-	¢D, 1	24.0	23.3	-	-	-	-
Pancress	-	40, 1	•	23.3	•	-		-
Lung	146.0 ^C	280.8 ⁸	144.2	93.4	114.8	153.7	68.2	48,2
Prostate	36.5	80.2	_	70.0	-	_	12.8	32.1

a Significant difference compared to each control.

b Not significant compared to each control.

a Lung cancer rates combined for races algorithmently different from comparable rates of north and adjacent controls.

Table 10. Crude cancer death rates for selected sites in legales by ledisidual fract and matched controls, average about death rate per 180,000.

MATCH I

Cancer Site	2303	Index 2392	34 र नं .	Adjacent *	South Controls	Central Centrols	North Controls	
Ores	27.6	-	-	50.0	43	-	5.0	
Pancress		14.4	34.9	6.9	14.4	21.9	1	
Lung	-	68.3	-	27:8	8.2	拉馬	24.7	
Bress	-	19.4		6.8	24.7	31.3	24.7	
Cervix		19.4	_	13.9	6.2	3.1	5.0	

MATCH

Cancer Site	Index WF	23G1 BF	Adjecent WF	Controls BF	Contral WF	Controja SF	North (elovno: AB
Orași	Ţ	<u> </u>	Ţ -	22.9	· -	-	-	
Pancreas	-	-	-	45.8	37.7	-	17.4	-
Lung	39.3	-	25.0	45.8	-	49.2	17.4	-
Gress!	196.5		50.D	22.9	<u>.</u>	-	2 7.1	14.0
Cervix	1 -	34.4	75.0	22.9		-	-	14.0

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